RISK MANAGEMENT OF PASSIVE SMOKING AT WORK AND AT HOME

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I. INTRODUCTION

In 1986 The Surgeon General of the United States Public Health Service (PHS) concluded that "involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers."[1] Also in 1986, The National Research Council (NRC) of the National Academy of Sciences, a congressionally chartered private body established to further scientific knowledge and to advise the federal government on scientific issues stated that, "Considering the evidence as a whole, exposure to ETS increases the incidence of lung cancer in nonsmokers."[2] In 1991, the National Institute for Occupational Safety and Health (NIOSH) declared environmental tobacco smoke (ETS) to be a "potential occupational carcinogen," legal terminology for a substance capable of causing human cancer or reducing its latency period.[3] In 1992, a major report issued by the United States Environmental Protection Agency (EPA) proclaimed ETS to be a "known human carcinogen."[4] Based upon a review of the total weight of

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1. UNITED STATES DEPARTMENT OF HEALTH & HUMAN SERVICES, THE HEALTH CONSEQUENCES OF INVOLUNTARY SMOKING 13 (1986) [*hereinafter* SURGEON GENERAL REPORT].

2. NATIONAL RESEARCH COUNCIL, BOARD ON ENVIRONMENTAL STUDIES AND TOXICOLOGY, COMMITTEE ON PASSIVE SMOKING, ENVIRONMENTAL TOBACCO SMOKE: MEASURING EXPOSURES AND ASSESSING HEALTH EFFECTS 10 (1986) [*hereinafter* NRC STUDY].

3. NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH, ENVIRONMENTAL TOBACCO SMOKE IN THE WORKPLACE, LUNG CANCER AND OTHER HEALTH EFFECTS 12 CURRENT INTELLIGENCE BULLETIN 54 (1991).

4. ENVIRONMENTAL PROTECTION AGENCY, RESPIRATORY HEALTH EFFECTS

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evidence, EPA concluded that ETS is a human lung carcinogen.[5] Moreover, in 1986, the NRC stated that because parental smoking increases pulmonary symptoms and respiratory infections, "it is prudent to eliminate ETS exposure from the environments of small children;"[6] the PHS also warned parents "to refrain from smoking in the presence of children."[7] In 1992 the EPA concluded that ETS exposure causes annually up to three hundred thousand cases of bronchitis and pneumonia in children up to eighteen months of age, causes increased prevalence of fluid in the middle ear, symptoms of upper respiratory tract infection, and a small but significant reduction in lung function.[8] EPA also concluded that ETS causes increased frequency of asthma, and aggravates the severity of asthma in up to one million children per year, and is a risk factor for asthma induction.[9] Further, in 1992, the American Heart Association declared ETS to be a "major preventable cause of cardiovascular disease and death," and estimated ETS-related mortality from heart disease to approach forty-five thousand annually.[10]

In 1985, the author applied risk assessment methodology to estimate the cancer risk to nonsmokers from environmental tobacco smoke in the workplace.[11] The study estimated that there were five thousand lung cancer deaths per year of nonsmokers in this country from passive smoking, and further predicted that the bulk of these deaths were due to workplace smoking.[12] These estimates were consistent with nine out of ten of the lung cancer risk assessments pub-

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OF PASSIVE SMOKING: LUNG CANCER AND OTHER DISORDERS 1-4 (1992) [*hereinafter* EPA REPORT].

5. This evidence includes animal bioassays and genotoxicity studies, biological measurements of human uptake of tobacco combustion products, the human epidemiological data on lung cancer induction at actual environmental levels of exposure to ETS, and the conclusive evidence of dose-response in active smokers, coupled with the chemical similarity of mainstream smoke and ETS. *Id.*

6. NRC STUDY, *supra* note 2, at 9.

7. *Id.* at xi.

8. EPA REPORT, *supra* note 4, at 1-5.

9. *Id.*

10. TAYLOR ET AL., AMERICAN HEART ASSOCIATION 86 POSITION STATEMENT: ENVIRONMENTAL TOBACCO SMOKE AND CARDIOVASCULAR DISEASE 1-4 (1992).

11. Repace & Lowrey, *A Quantitative Estimate of Nonsmokers' Lung Cancer Risk from Passive Smoking*, 22 ENVIRONMENT INT'L 3-23 (1985) [*hereinafter* Repace & Lowrey, *Quantitative Estimate*]; Repace & Lowrey, *An Indoor Air Quality Standard for Ambient Tobacco Smoke Based on Carcinogenic Risk*, 85 NEW YORK STATE J. MED. 381-383 (1985) [*hereinafter*, Repace & Lowrey, *Indoor Air Quality*].

12. Repace & Lowrey, *Risk Assessment Methodologies for Passive Smoking Induced Lung Cancer*, 10 RISK ANALYSIS 27, 34 (1990) [*hereinafter* Repace & Lowrey, *Risk Assessment Methodologies*].

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lished during the 1980's, which averaged 5000 plus or minus 2500 lung cancer deaths per year.[13] These estimates are consistent with the 1992 ETS risk assessment of the EPA, which conservatively estimated three thousand lung cancer deaths per year from passive smoking, and which also concluded that the bulk of those deaths were due to workplace exposures.[14] To place such estimates into perspective, environmental tobacco smoke is a greater risk to the public health of nonsmokers than all other hazardous industrial air pollutants regulated by the U.S. Environmental Protection Agency combined, including airborne radionuclides, arsenic, asbestos, benzene, coke oven emissions, and vinyl chloride.[15]

Exposures to ETS can be assessed by personal air contaminant monitoring, modeling of concentrations based upon a combination of air sampling, time-activity patterns, and questionnaires, or upon biological markers.[16] The author alone[17] and with another[18] published models capable of predicting levels of nonsmokers' exposure to atmospheric ETS nicotine, carbon monoxide, and respirable particles (RSP), and cotinine in body fluids. Such models enable calculation of nonsmokers' passive smoking risks from ETS RSP, ETS nicotine, or serum or urinary cotinine.[19]

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13. *Id.* at 27.

14. EPA REPORT, *supra* note 4, at 1-12.

15. Repace & Lowrey, *supra* note 12, at 27.

16. The two best methods at present are (1) based upon the biological markers, nicotine and its metabolite, cotinine, both of which are highly specific for ETS, and (2) based upon atmospheric nicotine from the gas phase of ETS, and respirable suspended particles (RSP) from the particulate phase of ETS, which is the largest fraction of ETS by weight. *See* SURGEON GENERAL REPORT, *supra* note 1, ch. 3; NRC STUDY, *supra* note 2, ch.5; EPA REPORT, *supra* note 4, at ch. 3.

17. J. Repace, *Concentrations of Environmental Tobacco Smoke: Effects of Ventilation and Room Size*, 9 INTERNATIONAL AGENCY FOR RESEARCH ON CANCER. PASSIVE SMOKING, ENVIRONMENTAL CARCINOGENS: SELECTED METHODS OF ANALYSIS, SCIENTIFIC PUBLICATION NO. 81, ch. 3 (1987) [*hereinafter* Repace, *Effects of Ventilation*]; J. Repace, *Indoor Concentrations of Environmental Tobacco Smoke: Field Surveys*, INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, PASSIVE SMOKING, ENVIRONMENTAL CARCINOGENS: SELECTED METHODS OF ANALYSIS, SCIENTIFIC PUBLICATION NO. 81, ch. 10 (1987) [*hereinafter* Repace, *Field Surveys*]; J. Repace, *Tobacco Smoke Pollution*, in NICOTINE ADDICTION ch. 7 (1993) [*hereinafter* Repace, *Tobacco Smoke Pollution*].

18. Repace & Lowery, *Quantitative Estimate*, *supra* note 11, at 13; Repace & Lowrey, *Indoor Air Quality*, *supra*, note 11 at 382; Repace & Lowrey, *Tobacco Smoke, Ventilation, and Indoor Air Quality*, 88 ASHRAE TRANSACTIONS 895-914 (1982); Repace & Lowrey, *Indoor Air Pollution, Tobacco Smoke, and Public Health*, 208 SCIENCE 464-474 (1980) [*hereinafter* Repace & Lowrey, *Public Health*].

19. Repace & Lowrey, *Quantitative Estimate*, *supra* note 11, at 3; Repace & Lowrey, *Indoor Air Quality*, *supra* note 11, at 383; Repace & Lowrey, *An*

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Further, assessment of individual building-specific risks of ETS-induced diseases can be performed, and the effect of physical arrangements common to various smoking policies or engineering control measures such as ventilation or air cleaning can also be evaluated.[20]

II. Determinants Of ETS Concentrations In Buildings

There are two major sources of exposure to ETS: the home and the workplace, in which 88% of the average American's daily activities take place.[21] As Figure 1 illustrates, under typical conditions of smoking occupancy and ventilation, buildings are heavily polluted with ETS-RSP.

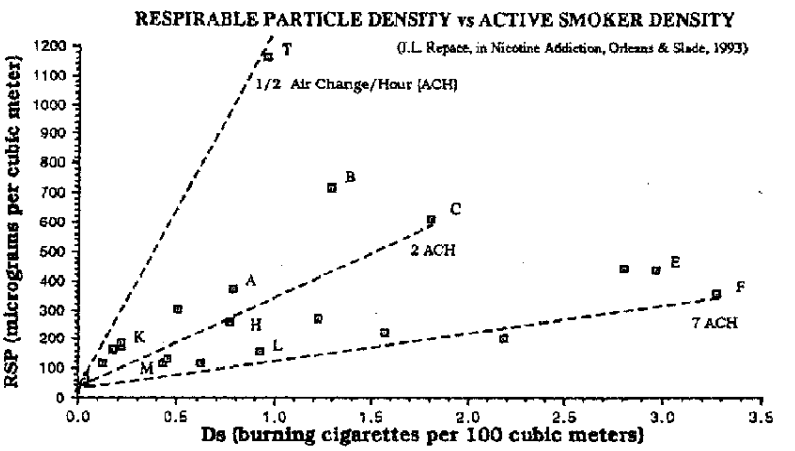

Fig. 1. RSP air pollution measured in 1 residential (A) and 17 commercial (B-T) buildings as a function of active smoker density Ds and air exchange rate Cv, in air changes per hour.[22]

Enforceable Indoor Air Quality Standard for Environmental Tobacco Smoke in the Workplace, 13 RISK ANALYSIS 463 (1993) [*hereinafter* Repace & Lowrey, *Enforceable Standards*].

20. *See supra* notes 17, 18, 19.

21. *See* Repace & Lowrey, *Quantitative Estimate*, *supra* note 11, at 5.

22. The mean level is about two hundred fifty micrograms per cubic meter of air ($\mu g/m^3$). By comparison, levels in thirty-three nonsmoking residential, commercial, and transportation microenvironments (at $D_s = 0$), showed a mean value of about 40 $\mu g/m^3$. Repace, *Field Surveys*, *supra* note 17, at 156-157; Repace, *Tobacco Smoke Pollution*, *supra* note 17, at 132.

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Figure 1 shows that ETS concentrations in both residential and commercial buildings (including bowling alleys, bingo parlors, restaurants, dinner theatres, bars, cocktail lounges, lodge halls, weddings, hospital waiting rooms, and sports arenas) are directly proportional to the smoker density (i.e., the number of smokers per unit volume) and inversely proportional to the air exchange rate. ETS RSP levels in a five hundred square foot office building conference room ventilated at the rate of four air changes per hour (ACH) with four chainsmokers were measured at nearly two thousand micrograms per cubic meter ($\mu g/m^3$), whereas background RSP levels would typically be 20 $\mu g/m^3$.[23]

Since building ventilation rates are tied to building occupancies and are code-specified, this means that nonsmokers' ETS exposures and doses are basically determined by the smoking prevalence among the building occupants and by the smoking policy. More than four hundred thousand tons of tobacco products are burned indoors each year, presenting overwhelming burdens to building ventilation systems which are designed to control carbon dioxide from human metabolism, not clouds of tobacco carcinogens and respiratory toxins.[24] For example, taking a high-rise building of five hundred thousand square feet containing four thousand workers, at a 25% smoking prevalence, approximately one to one and a half tons of carcinogenic tobacco tar from the smoking of approximately sixty-four million cigarettes will be liberated in the building over a twenty-year period.[25] Much of this will be absorbed onto the building surfaces where it will outgas, exposing nonsmokers to carcinogens, toxins, and odors long after smoking has ceased.[26] Air exchange rates in commercial buildings may be as low as 0.3 air changes per hour (ACH) in poorly ventilated office buildings to as high as 7 ACH in well-ventilated restaurants.[27] Open-window air exchange rates in naturally ventilated commercial and institutional buildings may range from 5 to 15 ACH in the mild season.[28] In a typical home, an average smoking homemaker will smoke about eleven

23. Repace & Lowrey, *Public Health*, *supra* note 18, at 466.

24. Repace & Lowrey, *Risk Assessment Methodologies*, *supra* note 12; Repace & Lowrey, *Public Health*, *supra* note 18.

25. *Id.*

26. NRC STUDY, *supra* note 2.

27. *Ventilation For Acceptable Indoor Air Quality*, ASHRAE Standard 62-1989.

28. Lowrey ET AL., *Indoor and Outdoor Respirable Suspended Particulates in Budapest, Hungary*, 3 PROCEEDINGS OF THE 6TH INT'L CONFERENCE ON INDOOR AIR POLLUTION 93-96 (1993) [*hereinafter* Lowrey, *Respirable Suspended Particles*].

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thousand cigarettes per year.[29] Closed-window annual average air exchange rates in homes may vary from as low as 0.1 ACH in super energy-efficient homes, to 1.5 ACH in poorly insulated ones.[30] Recent preliminary data from eight homes in Pennsylvania during the summer showed average air exchange rates of about 2 to 3 ACH in the non-air-conditioned homes, reflecting the effect of opened windows, compared to 0.3 to 0.4 ACH in the air-conditoned homes.[31]

A. *ETS In The Home*

In single family homes, twenty-four hour average personal exposures to RSP (particles two and a half micrometers in diameter or less) for ETS-exposed persons in twenty-four Vermont residences during the heating season were shown to be increased an average of 18 $\mu g/m^3$ relative to non-ETS exposed persons.[32] In similar studies, peak personal exposures were found to be more than ten times higher.[33] More recently, an EPA study of inhalable particulate matter of ten micrometers or less (PM_{10}), found a 33 $\mu g/m^3$ increase in nighttime average personal exposure for a statistical sample of one hundred seventy-eight nonsmoking residents of the City of Riverside, California, who resided with smokers relative to nonsmokers in nonsmoking homes.[34] These measured values are averaged over periods of nonsmoking.[35] Assuming smoking takes place only for six hours per day, in keeping with time-activity pattern studies, this suggests the average Vermont levels during smoking were 72 $\mu g/m^3$ RSP, and that the average California levels during smoking were 66 $\mu g/m^3$ PM_{10}. Since people vary in their activity patterns, and interior doors may be open or closed, the dilution volume of the space where ETS exposure might have occurred may be assumed to vary from a small one hundred square foot den through the eight hundred square foot area of a two bedroom apartment up to the entire fifteen hundred square feet of a typical single-family home (an eight foot ceiling is assumed).

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29. Repace & Lowrey, *Quantitative Estimate, supra* note 11.

30. J. Repace, *Indoor Air Pollution*, 8 ENVIRONMENTAL INT'L 21 (1982) [*hereinafter* Repace, *Indoor Air Pollution*].

31. H.H. Suh ET AL., *Indoor and Outdoor Acid Aerosol and Gas Concentrations*, 3 PROCEEDINGS OF THE 6TH INT'L CONFERENCE ON INDOOR AIR POLLUTION 251-256 (1993) [*hereinafter* Suh ET AL.].

32. Sexton ET AL., *Personal Exposure to Respirable Particles: A Case Study in Waterbury, Vermont*, 18 ATMOS. ENV. 1385-1398 (1984).

33. SURGEON GENERAL REPORT, *supra* note 1.

34. E.D. Pellizzari ET AL., *Particle Total Exposure Assessment Methodology (PTEAM)*, 1 RIVERSIDE, CALIFORNIA PILOT STUDY (1993).

35. *Id.*

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Figure 2 shows a plot of predicted ETS-RSP levels during smoking as a function of residential floor area for a single habitual smoker smoking at the U.S. average rate of two cigarettes per hour, assuming an emission of twenty-four milligrams of respirable particles per cigarette.[36]

Therefore, average ETS particle levels of this magnitude, used as a surrogate for all ETS, must be associated with the average observed increased risk of lung cancer in adults and respiratory disease in children.

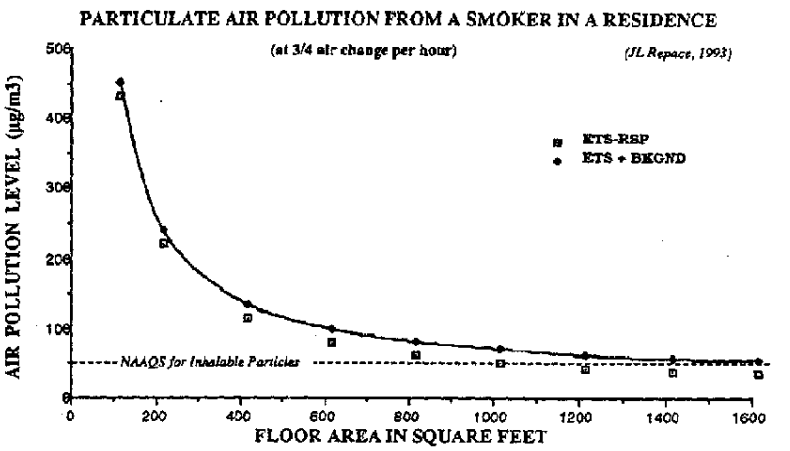

Fig. 2: Steady-state respirable particulate air pollution from ETS modeled during smoking from a single smoker in a residence (eight foot ceiling) with an average air exchange rate, as a function of residential floor area.[37]

36. The equation used to calculate the concentrations is $R1 = 72\ D_{hs}/C_v$ where D_{hs} is the smoker density in units of habitual smoker per 100 m^3, and C_v is the average household air exchange rate in units of air changes per hour (ACH) and assumes an average residential air exchange rate of 3/4 ACH. Repace, *Effects of Ventilation, supra* note 17.

37. ETS RSP is shown by itself and added to a typical 20 $\mu g/m^3$ background. When the exposure venue is small, levels of air pollution may approach those found in bars (C,F, & Q in Fig. 1). The average U.S. single family home has a floor area of fifteen hundred square feet.

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Figure 2 shows that for an area of about six hundred to eight hundred square feet comparable to the floor area of a one to two bedroom apartment, or the non-sleeping areas of a single-family home, the particulate air pollution levels due to ETS calculated for Waterbury, Vermont and Riverside, California during smoking are consistent with levels predicted by the ETS model of Repace[38]. The National Ambient Air Quality Standard (NAAQS) for PM_{10}, which does not take into account the chemical nature of the aerosol[39], is shown for comparison. It is seen that smoking in small space volumes, such as an enclosed recreation room, can increase the ETS concentration greatly. Moreover, a recent study of one hundred forty Suffolk and Onondaga County, New York homes, showed that cigarettes were a many times greater source of fine-particle air pollution than kerosene heaters (x3), woodburning (x6), and all other miscellaneous sources (x2).[40]

Figure 3 shows a plot of the eleven United States epidemiological studies of passive smoking and lung cancer considered by the EPA, which examined the risk of lung cancer in the nonsmoking spouses of smokers.[41] The exposure assessment technique compares more exposed to less exposed nonsmokers, not exposed to unexposed.[42] Nevertheless the increased risk is apparent in that eight of the eleven studies are positive.[43] The horizontal dashed line labelled "R&L Theory" shows that the lung cancer and passive smoking model of Repace & Lowrey predicts the pooled risk (labelled "All") exactly.[44] The "odds ratio" refers to the risk of ETS-induced lung cancer for nonsmoking women whose husbands smoke, relative to those whose husbands are nonsmokers.[45]

38. J. Repace, *Effect of Ventilation*, *supra* note 17 at 37.
39. J. Repace, *Field Surveys*, *supra* note 17, at 143.
40. P. Koutrakis ET AL., *Source Apportionment of Indoor Aerosols in Suffolk and Onondaga Counties, N.Y.*, 26 ENVIRON. SCI. TECH. 521-527 (1992).
41. EPA STUDY, *supra* note 4.
42. *Id.*
43. *Id.*
44. Repace & Lowrey, *Predicting the Lung Cancer Risk From Domestic Passive Smoking*, 36 AM. REV. OF RESPIRATORY DISEASE 1308 (1987) [*hereinafter* Repace & Lowrey, *Predicting Lung Cancer Risk*].
45. EPA STUDY, *supra* note 4.

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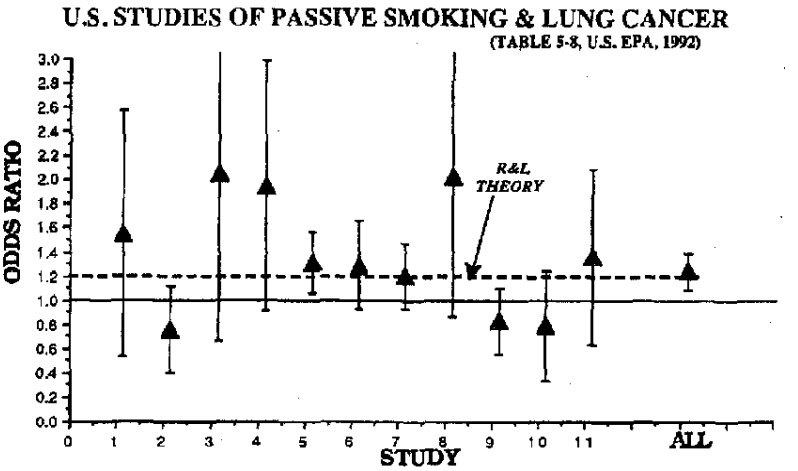

Fig. 3. Increased risk of lung cancer for 11 studies of U.S. nonsmoking women whose husbands smoke relative to those whose husbands are nonsmokers.[46]

B. *ETS At Work*

Dilution ventilation is the only basic ETS removal mechanism in most buildings.[47] In workplaces throughout North America, design ventilation standards are set by the American Society of Heating, Refrigeration and Ventilation Engineers (ASHRAE) under ASHRAE Standard 62, and implemented by local building codes in use throughout the United States.[48] The most recent version of this

46. The odds ratio was corrected by the EPA for smoker misclassification. The predictions of the Repace-Lowrey model, an odds ratio of 1.2, are shown for comparison. Repace & Lowrey, *Predicting Lung Cancer Risk*, *supra* note 44 at 1308.

47. Repace & Lowrey, *Quantitative Estimate*, *supra* note 11.

48. *Ventilation for Acceptable Indoor Air Quality*, ASHRAE STANDARD 62-1989 (1989).

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standard, ASHRAE 62-1989, assumes that the spacial occupancy of the workplace is seven workers per thousand square feet of floor area.[49] This standard recommends amounts of ventilation based upon occupancy, and provides for twenty cubic feet per minute of outdoor ventilation air per occupant (CFM/OCC) in offices "with a moderate amount of smoking".[50] This corresponded to one-third of the workers smoking cigarettes (typical at the time the standard was drafted) at the "moderate" rate of two per hour.[51] Higher rates of ventilation, 60 CFM/OCC, are prescribed for smoking lounges.[52] These recommended rates are specified to limit the unacceptablity of the *odor* of ETS.[53] This so-called "ventilation rate procedure," which the tobacco industry promotes (implicitly) as a health-based standard for ETS, is *not* designed to limit human *health risk* from ETS.[54] ASHRAE 62-1989 actually specifies that "If human carcinogens or other harmful contaminants are suspected to be present in the occupied space, other relevant standards or guidelines . . . must supersede the ventilation rate procedure."[55] In 1983, the author observed that for workplaces ventilated over the entire range of ASHRAE-specified ventilation rates, from 5 CFM/OCC (designed for energy conservation, to 50 CFM/OCC, the lung cancer risk from forty years' exposure (a working lifetime) to passive smoking ranged from ten excess lung cancer deaths (LCDs) per thousand workers to about one LCD per thousand workers.[56] This calculation was based upon a 33% smoking prevalence, and ASHRAE Standard 62-1981 for building occupancy. ASHRAE 62-1989 did not recognize that ETS is a human carcinogen due to the threat of tobacco industry litigation.[57] Thus, despite claims to the contrary by the tobacco industry that ASHRAE standards are designed to control tobacco smoke,[58] ASHRAE-recom-

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49. *See* Table 2.
50. *Id.*
51. This is actually the average United States rate, whereas chain smokers may smoke at triple the average. Repace & Lowrey, *Quantitative Estimate, supra* note 11.
52. ASHRAE STANDARD 62-1989; *see* Table 2, at 8-9.
53. *Id.*
54. *Id.*
55. *Id.*
56. J. Repace, *Effect of Ventilation on Passive Smoking Risk in a Model Workplace*, MANAGEMENT OF ATMOSPHERES IN TIGHTLY ENCLOSED SPACES (American Society of Heating Refrigerating and Airconditioning Engineers (ASHRAE) eds. 1983).
57. J. Repace, *The Dramatic Changes in Recommended Outdoor Air Rates—What Are the Rational Motives and Consequences?* 4 CONCLUSIONS AND RECOMMENDATIONS FOR HEALTHIER BUILDINGS '88 132 (1991).
58. Repace & Lowrey, *Issues and Answers on Passive Smoking in the Workplace: Rebutting Tobacco Industry Arguments*, 1 TOBACCO CONTROL INT'L

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mended ventilation rates correspond to cancer risks which are more than one thousand times the *de minimis* level, even at the current national smoking prevalence of 25%.[59] Therefore, the ventilation rate procedure is inoperative, and other health-based standards for ETS must be used.[60]

III. HEALTH-BASED STANDARDS FOR ETS

To assess the risk of passive smoking, the methodology of *quantitative risk assessment*, originally developed by physicists to assess the cancer risks of nuclear power, must be used[61]. Risk assessment is routinely used by United States federal regulatory agencies such as the EPA, Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA), to assess the carcinogenic risks of radiation, chemicals, and biological substances, in air, water, and food, e.g., radon gas from uranium mines, halomethanes in chlorinated drinking water, and aflatoxin B1 in peanut butter.[62] The gravity of the cancer risk is judged by how far the risk is above the "acceptable" or *de minimis*, risk level.[63] This level is actually a range which varies from a lifetime probability of cancer death of one death in a population of ten million at risk to one death per one hundred thousand, depending upon the regulatory agency, and the size of the population at risk.[64] In the case of large populations at risk of cancer, a review of one hundred thirty-two federal regulatory decisions disclosed that a *de minimis* level of one lifetime death per million population at risk is generally used by federal agencies,[65] although when the carcinogen is in food or drinking water, a one in ten million risk level is often used.[66] The appropriate *de minimis* risk level for ETS is determined from consideration of the size of the population at risk.[67] The EPA, in setting its National

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208-219 (1992) [*hereinafter* Repace & Lowrey, *Rebutting Industry Arguments*].

59. Unpublished data on file with Office on Smoking and Health.

60. Repace & Lowrey, *Indoor Air Quality, supra* note 11; Repace & Lowrey, *Enforceable Standards, supra* note 19.

61. NATIONAL ACADEMY OF SCIENCES, RISK ASSESSMENT IN THE FEDERAL GOVERNMENT, MANAGING THE PROCESS (1983).

62. *Id.*

63. *Id.*

64. E.L. Anderson, ET AL., *Quantitative Approaches in Use to Assess Cancer Risk*, 3 J. RISK ANALYSIS 277-295 (1983).

65. C.C. Travis ET AL., *Cancer Risk Management*, 21 ENVIRONMENTAL SCIENCE AND TECHNOLOGY 415-420 (1990) [*hereinafter* Travis, *Risk Management*].

66. *Perrier Recalls Bottled Water*, NEW YORK TIMES, February 11, 1990.

67. Travis, *Risk Management, supra* note 65.

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Emission Standards for Hazardous Air Pollutants[68] has regulated pollutants to levels which had national impacts after control of less than one death per year. The population at greatest risk from ETS is the twenty-eight and a half million nonsmoking workers who in 1988 were employed in workplaces with few or no restrictions on smoking.[69] For a working-lifetime of forty years, this results in an annual risk of 2.5×10^{-8}. When multiplied by the population at risk, this yields less than one death per year in the population at greatest risk, after control.[70] To help place the *de minimis* risk concept into further perspective, consider the recent experience involving Perrier mineral water.[71] In 1990, Perrier Corporation recalled its entire worldwide stock of bottled water because of benzene contamination, at a cost of over seventy million dollars in the United States alone.[72] Perrier's recall was prompted by an FDA risk assessment which showed that the lifetime risk of cancer from drinking two bottles of Perrier per day was one death per million exposed.[73]

An ETS risk assessment methodology was developed in 1985 correlating a working lifetime lung cancer risk of one per hundred thousand (10^{-5}) to an average ETS RSP level of 0.75 micrograms per cubic meter of workplace air.[74] This methodology was utilized in a 1989 Department of Transportation study of airliner cabin air quality, for estimating the risk of passive smoking to crew and passengers,[75] and in testimony by the author before the 1989 House Aviation Subcommittee in hearings which resulted in a smoking ban on U.S. domestic flights.[76]

We now examine the risks to nonsmokers under five types of smoking policy: 1. No restrictions; 2. Spacial separation of smokers from nonsmokers; 3. Physical separation of smokers and nonsmokers on the same ventilation system; 4. Physical separation on different ventilation systems; 5. A ban on indoor smoking.

For any particular building, the calculation can be tailored to the local conditions. Nonsmokers' lung cancer risk from passive

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68. *See* 42 U.S.C. § 7412 (1992).
69. Repace & Lowrey, *Indoor Air Quality, supra* note 11.
70. Repace & Lowrey, *Risk Assessment Methodologies, supra* note 12.
71. *Perrier Recalls Bottled Water*, N.Y. TIMES, Feb. 11, 1990.
72. *Id.*
73. *Id.*
74. Repace & Lowrey, *Quantitative Estimate, supra*, note 11, at 3; Repace & Lowrey, *Indoor Air Quality, supra* note 11, at 382.
75. U.S. Dept. of Transportation, N.L. Nagda ET AL., *Airliner Cabin Environment: Contaminant Measurements, Health Risks and Mitigation Options* (1989).
76. *Hearings Before the Aviation Subcomittee*, U.S. House of Representatives, 100th Cong. (July 1989).

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smoking in a model office workplace as a function of ventilation rate is illustrated in Figure 4, referred to the one-per-million *de minimis* risk level.

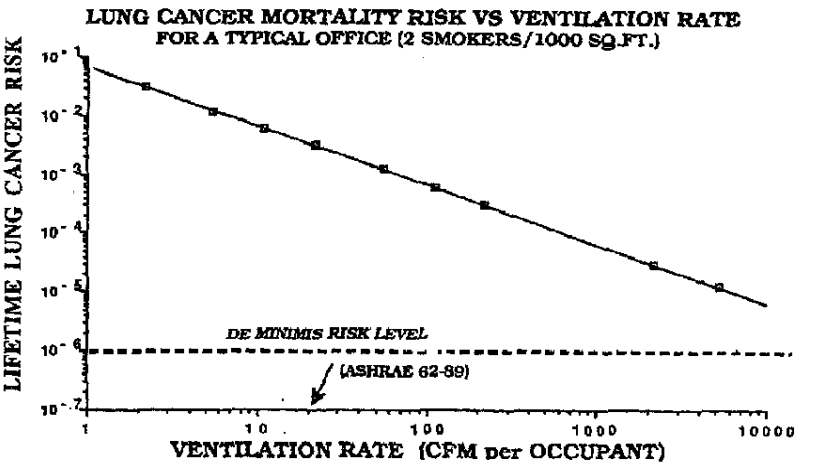

Fig. 4. Estimated working-lifetime lung cancer mortality risk from ETS exposure in an office vs. outdoor air ventilation rate.[77]

The risk level is given in units of lung cancer deaths per working lifetime (forty years) by the formula: RISK = {2.89 x 10-3 D_{hs}/C_v}.[78]

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77. Attainment of *de minimis* risk using ventilation or air cleaning is impossible, as it would require thousand-fold increases over building code-specified ventilation rates. The ASHRAE Standard of 20 CFM/OCC corresponds to an air change rate of 0.84 air changes/hr (ACH). Repace & Lowrey, *Indoor Air Quality*, *supra* note 11.

78. Where D_{hs} is the habitual smoker density in units of habitual smokers per hundred cubic meters of building volume, and C_v is the outdoor air supply rate units of air changes per hour. At a 29% smoking prevalence and an occupancy of seven persons per thousand square feet, there is an occupancy of about two habitual smokers per thousand square feet (assuming a ten foot ceiling), and D_{hs} = 0.72 habitual smokers per hundred cubic meters, and at the ASHRAE

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IV. Risk Management Strategies In Use For ETS In Buildings.

How do ventilation, air cleaning, separating smokers from nonsmokers within a room or on separate ventilation systems, and smoke-free buildings compare as risk management strategies for ETS?

A. *Ventilation*

Contrary to the tobacco industry's claims, Figure 4 shows that under ASHRAE standard 62-1989, the lung cancer risk to nonsmoking workers is between two and three per thousand, or 2000x to 3000x the *de minimis* level.[79] From Figure 4, the effect of increases in ventilation on ETS risk in a workplace with unrestricted smoking can also be evaluated. Although the current ASHRAE Standard mandates ventilation rates of 20 CFM/OCC in office buildings with smoking[80], this mandate is often observed in the breach, with the desire of building owners to reduce costs often exceeding their desire to obey the building code, and ventilation rates of 5 CFM/OCC designed to conserve energy have persisted.[81] However, even if building owners could be persuaded to increase ventilation rates to control smoking, as Figure 4 shows, more than a thousand-fold increase in ventilation rate is required to attain *de minimis* risk at typical smoking rates. Such impractical increases in ventilation rate would result in a virtual windstorm indoors. Increases in ventilation rate by more than a factor of two would likely require replacing the ventilation system.[82] Although in homes, ventilation rates can be increased by opening windows and using exhaust fans, during the heating and cooling seasons these methods are unlikely to be used.

B. *Air Cleaning*

Although some air cleaners such as electrostatic precipitators (ESPs) and high-efficiency particle (HEPA) bag filters may have high efficiencies for fine particles, they must process many room volumes

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ventilation rate of twenty cubic feet per minute per occupant, C_v = 0.84 air changes per hour, and RISK = 2.5 x 10^{-3}, or two hundred fifty lung cancer deaths per one hundred thousand workers at risk per forty-year working lifetime. Repace & Lowrey, *Indoor Air Quality*, *supra* note 11.

79. Repace & Lowrey, *Rebutting Industry Arguments*, *supra*, note 58.
80. Repace & Lowrey, *Indoor Air Quality*, *supra* note 11.
81. *Id.*
82. ASHRAE, ASHRAE HANDBOOK OF FUNDAMENTALS (1993).

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of air per hour to remove particles significantly.[83] Moreover, ESPs and HEPAs do not remove gases, and many of the carcinogens in ETS are in the gas phase, including the bulk of what was originally in the carcinogenic particulate phase as the tobacco aerosol ages.[84] Although combination charcoal-purafil gas-removal coupled with HEPA filters are effective on both gas and particulate phase ETS, they are limited by the unattainable requirement of processing two thousand two hundred sixty room air volumes per hour to reach a one per one million risk.[85] In order to attain *de minimis* risk by using air cleaning to control ETS, an estimated capital expenditure of two hundred thousand dollars per smoker (in 1984 dollars) would be required exclusive of maintainance.[86] In other words, reducing levels of a dilute pollutant in a large dilution volume is futile, due to the laws of physics. Also, when charcoal filters become saturated, they are subject to "break-thru" of contaminants and thus, filters must be rigorously changed.[87] A more fundamental limitation is that no filter can remove ETS on its way past the nonsmoker's nose to the air cleaner. Figure 4 also applies to air cleaning. Thus, air cleaning — like filtration of a lake to remove water pollutants — is a futile exercise to control ETS. The type of air cleaners used in homes are typically inefficient for tobacco smoke-sized particles because high capture efficiencies for small particles come at high cost which the market will not bear.[88]

C. *Smoking Sections*

Smoking sections within a space have often been used in restaurants, aircraft cabins, and in many workplaces.[89] As Figure 5 shows, levels of ETS are indeed higher in the smoking section than in the non-smoking section.[90] However, confining smoking to one side of a room does not reduce the ETS loading of the space.[91] Thus, tobacco smoke will rapidly diffuse throughout the space and

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83. *Id.*
84. *Id.*
85. *Id.*
86. *Id.*
87. EPA REPORT, *supra* note 4.
88. P. McNall, 30 ARCH. ENVIRON. HEALTH 552 (1975).
89. Repace & Lowrey, *Indoor Air Quality*, *supra* note 11.
90. Repace & Lowrey, *Ventilation and Indoor Air Quality*, *supra* note 18, at 910.
91. As any first-year student of the thermodynamics of gases knows, there is no "Maxwell's Demon" to keep ETS molecules in the smoking section from diffusing into the nonsmoking section by Brownian motion. *See e.g.*, E.U. CONDON & U. ODISHAW, HANDBOOK OF PHYSICS (1958).

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the laws of physics also rule out this putative control measure.[92] Moreover, smoking sections may increase risk for such workers as airline flight attendants and restaurant service personnel who must work in smoking sections.[93] The situation is similar for smoking in homes. Smoking in one room will rapidly diffuse into another unless its door is kept closed. It must be kept in mind that at an air exchange rate of 1 ACH, it takes more than three hours for 95% of the smoke in the air to dissipate once smoking is ended, and if the door is opened in that time, the remaining smoke will escape to the remainder of the house.[94]

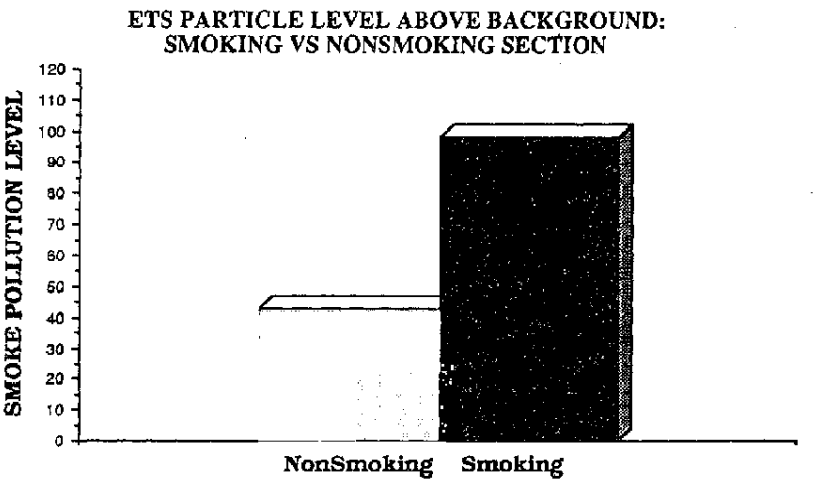

Fig. 5 An average of the ETS particulate level in micrograms per cubic meter (µg/m3) measured in three Washington DC area restaurants in the smoking and nonsmoking sections, above a non-ETS background RSP average of 25 µg/m3.[95]

92. *Id.*

93. J. Repace, *Are Indoor Air Quality Standards Needed?* 4 CONCLUSIONS AND RECOMMENDATIONS FOR HEALTHIER BUILDINGS '88 53 (1991).

94. G. Lofroth, TOBACCO CONTROL (forthcoming 1994)(on file with author).

95. Repace & Lowrey, *Ventilation and Indoor Air Quality, supra* note 18, at 910.

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D. *Separating Smokers In Different Rooms In The Same Building*

Separation of smokers from nonsmokers in physically separate spaces on the same ventilation system assumes — on faith — that the air volume of the building will dilute the risk down to an acceptable level. This "dilution-solution-to-pollution" method of control does not reduce the total ETS loading of the building.[96] As Figure 6 shows, under ASHRAE standard ventilation, more than one million square feet of building area *per smoker* are required to achieve *de minimis risk* — larger than most buildings.[97]

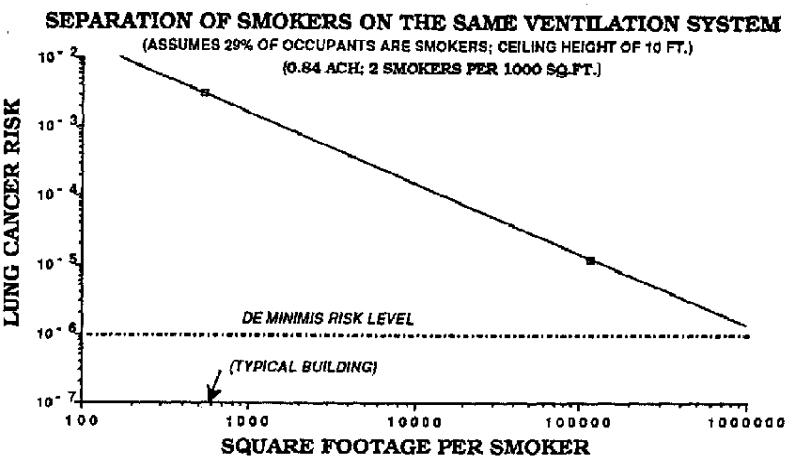

Fig. 6. Relying on the dilution volume of a building to achieve de minimis risk requires in excess of 1 million square feet of floor space per habitual smoker.

While such a policy may be an improvement over having smokers and nonsmokers in the same workroom, recirculation of tobacco smoke in central ventilation systems is an important source of exposure of nonsmokers to ETS in mechanically ventilated office

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96. The smokers are only re-arranged in the space, not restricted from smoking.

97. The median number of square feet per building for the four and one half million buildings in the United States is forty-five hundred square feet and the mean is fourteen thousand square feet. U.S. DEPT. OF COMMERCE, STATISTICAL ABSTRACTS OF THE UNITED STATES (1993).

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buildings.[98] "[C]entral air circulation systems and people movement increase the nicotine level throughout all rooms of a centrally ventilated building, regardless of the smoking policies of an individual office complex."[99] For example, restricting smoking in a highrise office building to the thirty-second floor snack bar resulted in reductions in ETS nicotine at nonsmokers' desks, which averaged 1 to 5 $\mu g/m^3$ prior to restrictions.[100] However, residual levels of nicotine at nonsmokers desks two months later remained at 0.4 $\mu g/m^3$ on the thirty-first floor, which was on the same ventilation system.[101] While such reductions ostensibly seem significant, the risk associated with such a residual level remains fifty times the *de minimis* risk level as shown in Figure 7.

In 1993, the author developed an indoor air quality standard based upon nicotine in workplace air, which for the first time, shows how to interpret nonsmokers' risks from second-hand smoke in individual workplaces from nicotine measurements in workplace air.[102] Analyzing published data from over two hundred workplaces, the authors of the study concluded that ETS typically poses levels of risk far beyond the so-called *de manifestis* risk level above which the federal government strictly regulates cancer-causing substances.[103] This was found to be true irrespective of whether the workplaces employed white collar, blue collar, or restaurant service workers.[104] A method was developed to analyze the levels of the nicotine metabolite, cotinine, in the body fluids of nonsmokers.[105] Based on data from four thousand persons, the levels of cotinine found in the blood and urine of typical nonsmokers indicate second-hand smoke lung cancer risks which are thousands of times the large-population *de mininimis* risk level for carcinogenic residues in air, water, or food.[106]

Figure 7 shows a plot of the lung cancer risk associated with various ETS nicotine levels in the workplace (eight-hour time-weight-

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98. D.C. Williams ET AL., *Measurement of Nicotine in Building Air as an Indicator of Environmental Tobacco Smoke Levels*, 60 ENVIRON. HEALTH PERSPECTIVES 405-410 (1985) [*hereinafter* Williams ET AL.]; W.M. Vaughn & S.K. Hammond, *Impact of "Designated Smoking Area" Policy on Nicotine Vapor and Particle Concentrations in a Modern Office Building*, 40 J. AIR WASTE MANAGE. ASSOC. 1012-1017 (1990) [*hereinafter* Vaugn & Hammond].
99. Williams ET AL., *supra* note 98.
100. Vaughn & Hammond, *supra* note 98.
101. *Id.*
102. Repace & Lowrey, *Enforceable Standards*, *supra* note 19, at 463.
103. *Id.*
104. *Id.* at 470.
105. *Id.* at 466.
106. *Id.* at 471.

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ed average (TWA)).

E. Smoking Lounges On Separate Ventilation Systems

Assuming the designated smoking areas are under an adequate amount of negative air pressure with respect to the nonsmoking areas of the building, such that no backstreaming of smoke can occur, proponents assert that this policy will reduce the risk of nonsmokers to zero. If nicotine tests show that these areas in fact do not leak, this is a completely adequate policy insofar as protecting nonsmokers.[107] Of course, the building owner must bear the expense of providing an area set aside strictly for smoking, ventilated at significantly higher rates, and adequately negatively pressurized with respect to the rest of the building, even under adverse wind and temperature conditions. Morover, the ventilation system must be balanced properly and maintained in balance throughout the life of the building. However, designated smoking areas that are separately ventilated and under negative pressure, may leak smoke into the rest of the building.[108] For example, smoking in a snack bar in a highrise office building on the thirty-second floor resulted in residual ETS nicotine at nonsmokers' desks on the nineteenth floor, which was not on the same air-handling system as the snack bar.[109] Thus it appears that permitting smoking in separately ventilated smoking lounges may not work very well in practice. Moreover, they cost far more than smoke-free buildings.[110]

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107. This statement remains true provided that nonsmokers do not enter the room.

108. F.A. Stillman, ET AL., *Ending Smoking at the Johns Hopkins Medical Institutions: An Evaluation of Smoking Prevalence and Indoor Air Pollution*, 264 JAMA 1565-1569 (1990) [*hereinafter* Stillman ET AL.]; L. Alivantes ET AL., *Tracer Gas Techniques for Measuring the Effectiveness of Pollutant Removal From Localized Sources*, PROC. IAQ 93 (1993); B. Ddworkis, *Measurement of Nicotine Leakage From Smoking Lounges* (unpublished manuscript on file with author).

109. Vaughn & Hammond, *supra* note 98.

110. Nonsmoking buildings require only the posting of signs.

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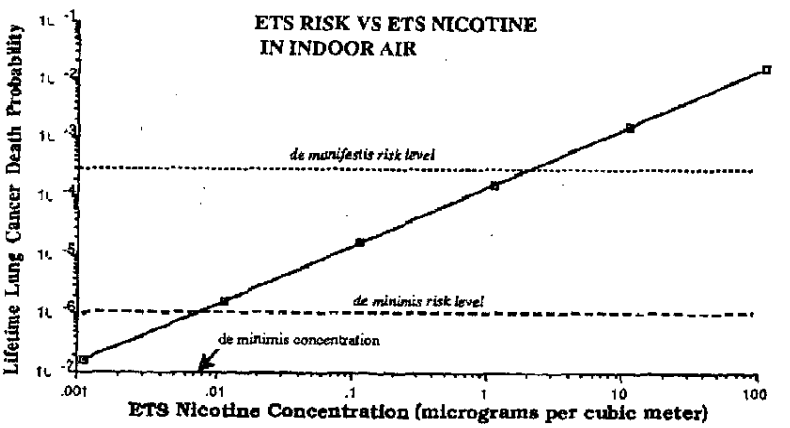

Fig. 7. Lifetime lung cancer risk associated with forty years workplace exposure to ETS at the associated ETS nicotine level (eight-hr TWA).[111]

Assuming an occupancy of thirty-five to fifty smokers in a well-designed thousand square foot smoking lounge, the capital cost to the building owner would be twenty-five to thirty thousand dollars to build, and the operating cost about twenty-four thousand dollars per year.[112]

Furthermore, a major problem remains due to the risk smokers have from each others' smoke.[113] Smokers appear to have a considerably increased risk of cancer from other smokers' ETS.[114] Relative to smokers who cohabited with nonsmokers, *smokers* who cohabited with one, two, or three or more other smokers had 40%, 120%, or 140% excess cancer risk, respectively[115], as illustrated in Figure 8. Since the risk of cancer (lung, throat, pancreas, etc.) for active smokers is already very considerable,[116] this policy of con-

111. Typical levels of nicotine in the workplace generally have been measured in the range one to one hundred micrograms per cubic meter. Repace & Lowrey, *Enforceable Standards, supra* note 19, at 472.

112. J. Poynter, *Take Ten Break Rooms* (1992) (unpublished manuscript on file with author).

113. Sandler ET AL., *Cumulative Effects of Lifetime Passive Smoking on Cancer Risk*, THE LANCET, 312-314 (Feb. 9, 1985) [*hereinafter* Sandler ET AL.].

114. *Id.*

115. *Id.*

116. *See*, U.S. DEPT. OF HEALTH AND HUMAN SERVICES, SMOKING AND HEALTH, 25 YEARS OF PROGRESS (1989).

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centrating smokers in a smoking area could result in considerably increasing the risk of an already extremely harmful habit. Furthermore, this policy is not as effective in enhancing quit-rates as smoke-free buildings, and as such would not be the choice of an employer who is interested in the wellness of all its employees, not just nonsmokers.

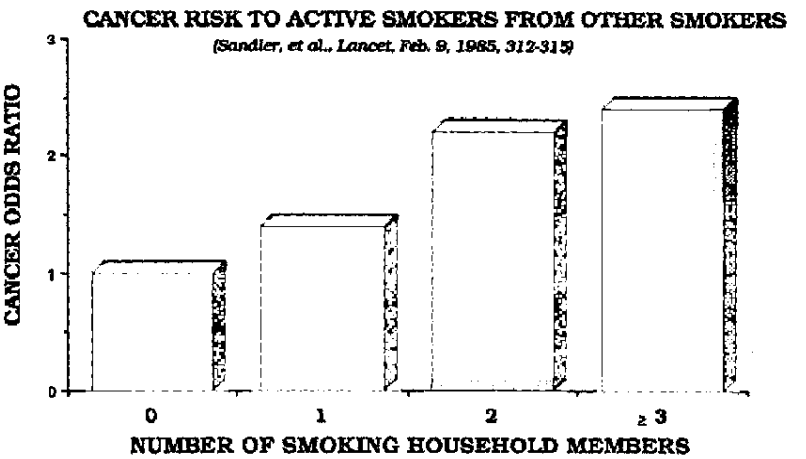

Fig. 8. Smokers who live with smokers suffer increased risk of cancer.[117]

F. Smoke-Free Buildings

This policy protects all workers from the harm of ETS equally, is the easiest to enforce, and represents the least cost, requiring only a twenty-five cent nonsmoking sign on each door. Smoke-free buildings have the additional considerable advantage of providing an incentive for smokers to quit. This is important to employers not only for humanitarian reasons, but also because of simple economics. Since smokers die on average seven years earlier than nonsmokers, have significantly higher rates of morbidity, absenteeism, and mortality than nonsmokers during their working years, smokers generally

117. Thus, smoking lounges in workplaces increase the risk of cancer among smokers, driving up morbidity and mortality which employers' health insurance plans must indemnify. Restricting smoking to outdoors avoids this problem. Sandler ET AL., *supra* note 113.

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cost employers more than nonsmokers.[118] Smoke-free workplaces increase one-year post-ban quit rates by up to 20%.[119] If smoke-free workplaces were employed on a national scale, this would result in a decrement in total emissions of ETS in all buildings of from fifty thousand to one hundred thousand tons per year.[120] Thus, smoke-free buildings, if applied nationally, make good pollution prevention policy.[121] Moreover, there does not appear to be any negative economic impact (except for the tobacco industry, perhaps). For example, in an examination of restaurant sales data, for fifteen cities in California and Colorado and over one hundred fifty-six sales quarters since 1992, no evidence was found that 100% smoke-free restaurant ordinances have any effect on sales.[122]

V. CONCLUSION

There is now a national consensus among U.S. environmental, occupational, public health, and scientific authorities that passive smoking, the breathing of environmental tobacco smoke, is a cause of respiratory disease, including lung cancer, in healthy adult nonsmokers, and of respiratory disease in children. This places ETS in the category of Hazardous Air Pollutants. Levels of ETS exposure and risk in any building, commercial or residential, can now be quantified using models or measurement of ETS tracers in the air or in body fluids. This quantification permits the evaluation of individual risks and benefits of various control measures.

Of all possible smoking policies the only one which will result in a acceptable risk to all persons, smokers as well as nonsmokers, from exposure to environmental tobacco smoke in buildings is one which restricts smoking completely to the outdoors. It is also the least costly policy to implement. Air pollution levels during smoking in the home exceed the level of the national ambient air quality standards for inhalable particulate matter on a simple mass concentration basis without even considering the chemical nature of the tobacco aerosol from ETS. During parental smoking in the home,

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118. M. Kristein, *Economic Issues Related to Smoking in the Workplace*, 89 NEW YORK STATE J. MED. 44-47 (1989); U.S. DEPT. OF HEALTH AND HUMAN SERVICES, SMOKING IN THE WORKPLACE (1985).

119. Stillman ET AL. *supra* note 108.

120. Repace & Lowrey, *Risk Assessment Methodologies*, *supra* note 12, at 28.

121. Repace, *Passive Smoking Has No Place in the Workplace*, 133 CANADIAN MED. ASSOC. J. 737-738 (1985).

122. Institute for Health Policy Studies, S.A. Glantz & L.R.A. Smith, THE EFFECT OF ORDINANCES REQUIRING SMOKEFREE RESTAURANTS ON RESTAURANT SALES (February 1994).

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